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Abstracts

Germ cells and gametogenesis

Program/Abstract # 269

LSD1 contributes to germline immortality in *C. elegans* by reprogramming epigenetic memory

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Upon their specification, the two primordial germ cells (PGCs) in *C. elegans*, Z2 and Z3, rapidly lose the euchromatic mark di-methylation of histone H3 on lysine 4 (H3K4me2). This epigenetic erasure has been suggested to contribute to embryonic germline maintenance through chromatin-based transcriptional repression. The mammalian protein LSD1 has been shown to demethylate H3K4me2. Here we show that mutants in the *C. elegans* LSD1 ortholog, spr-5, exhibit progressive sterility over many generations due to defects in oogenesis and a delay in spermatogenesis. The progressive sterility correlates with increased retention of H3K4me2 in the PGCs, suggesting that SPR-5 mediated H3K4 demethylation is essential for germline immortality. In addition, microarray analysis and quantitative rt-PCR in spr-5 mutants demonstrate that the progressive sterility corresponds with a heritable build up of expression in spermatogenesis genes. Taken together, these results suggest that failure to erase H3K4me2 in the germline cycle causes a progressive failure of epigenetic erasure in the PGCs which leads to inappropriate expression of spermatogenesis genes and increasing sterility. From these data we conclude that H3K4me2 can serve as an epigenetic memory and that LSD1 demethylases play a critical role in the reprogramming of this memory in the germline, preventing inappropriate epigenetic information from being propagated from one generation to the next.

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Program/Abstract # 270

The intriguing interaction of Dicer (DCR-1) with GLH-1, a P granule component in *Caenorhabditis elegans*

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The *G*erm*l*ine RNA *H*elicases (GLHs) are a family of four proteins that associate throughout all stages of development with the germline P granules of *Caenorhabditis elegans*. GLH-1 is essential for fertility, although how GLH-1 functions in germline development is still largely unknown. Through immunoprecipitations, we have recently determined that GLH-1 and DCR-1 bind one another and their interaction is not dependent upon RNA. Along with physical binding studies, we also identified a genetic interaction between GLH-1 and DCR-1. In the *dcr-1(ok247)* null mutant, GLH-1 levels are reduced or nearly absent, both by western blot analysis and by immunocytochemistry. Conversely, we find that DCR-1 protein levels are dramatically reduced in western blot analyses of *glh-1(gk100)*, a strong loss-of-function mutant, indicating that GLH-1 and DCR-1 levels are mutually dependent. How might the regulation of these two proteins be related? Microarray analysis of the *dcr-1(ok247)* mutant by the Bass laboratory recently reported that *glh-1* mRNA is significantly reduced when DCR-1 is missing; perhaps DCR-1 regulates *glh-1* mRNA levels through miRNAs. We also observed that DCR-1 protein levels increase within hours when worms are grown in JNK or proteosome inhibitors, implying that DCR-1 may be targeted for degradation by the Jun-terminal kinase KGB-1 (one of only three JNKs in *C. elegans*), as is the case for GLH-1. Based on our findings, we propose that GLH-1 may associate with DCR-1 to facilitate the miRNA pathway, directing the RISC complex to appropriate mRNA targets. These interactions could occur in the P granules or in the *C. elegans* germline RNA granules that are similar to somatic P bodies.

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Program/Abstract # 271

A dominant suppressor of the *fog-1*(*q253*ts) allele maps to *C. elegans LGII*

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Germ cell fate in *C. elegans* is determined through a complex genetic pathway ending in *fog-1* and *fog-3*. *fog-1* encodes a member of the *C*ytoplasmic *P*olyadenylation *E*lement *B*inding (CPEB) protein family. In other model systems, CPEB proteins have been shown to regulate translation of target messages. FOG-1 is required for germ cell proliferation and spermatogenesis in *C. elegans*; however its target messages and exact mechanism of activity in the worm germline remain to be elucidated. To identify proteins that either regulate or interact with FOG-1, we performed a genetic suppressor screen. We mutagenized approximately 85,000 haploid genomes and identified two dominant suppressors of the *fog-1*(*q253*ts) allele. We have mapped one of the dominant suppressors to *LGII* using genetic and snip-SNP mapping.

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Program/Abstract # 272

Regulation of the actin cytoskeleton during *Drosophila* oogenesis by Ena and Capping Protein

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